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# RODENTICIDE ECOTOXICOLOGY: PRE-LETHAL EFFECTS OF ANTICOAGULANTS ON RAT BEHAVIOUR

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**ABSTRACT:** Anticoagulant rodenticides may pose a secondary poisoning hazard to non-target predators and scavengers because of the time-delay between ingestion of a lethal dose and death of a target rodent. We investigated some pre-lethal effects of an anticoagulant rodenticide on the behaviour of wild rats in cages and in enclosures. We found that social interactions shortened time to death, that most rats died away from cover and that thigmotactic behaviour was reduced in the enclosures. The normal light-dark rhythm was upset in intoxicated rats in both cages and enclosures. Thus pre-lethal effects are likely to alter the exposure of predators and scavengers to intoxicated rats, and diurnal predators may be exposed more than nocturnal predators as a consequence. We stress the need to extend these behaviour studies to the field.

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## INTRODUCTION

When rodents have been poisoned with anticoagulant rodenticides, death does not occur immediately after ingestion. For example, time to death after consumption of a lethal dose of anticoagulant has been reported to range from four to nine days (Meehan 1984). Thus poisoned rats (*Rattus norvegicus*), and possibly other rodents, could represent a potential secondary poisoning hazard to scavengers and predators (Smith et al. 1990).

Laboratory trials in which predators have been fed on anticoagulant-poisoned carcasses have indicated a potential hazard (Savarie et al. 1979, Evans and Ward 1967, Mendenhall and Pank 1980). Kaukeinen (1982) acknowledged the limitations of such captive studies and suggested that rodents that have died from anticoagulants generally pose less hazard than active individuals with toxic residues in the days before death or recovery, as many predators of rodents do not prefer dead carcasses as prey items.

Townsend et al. (1981) looked at the possible effects of warfarin bait on woodmice (*Apodemus sylvaticus*) and concluded that tawny owls (*Strix aluco*) would be unlikely to take only contaminated prey. Townsend et al. (1984) carried out similar trials feeding warfarin-poisoned mice to least weasels (*Mustela nivalis*) and decided that weasels could be more at risk than tawny owls. Townsend et al. (1984) pointed to the need for field studies to see how likely it is that predators or scavengers would be exposed to enough poisoned prey items to cause death. The present study aims to consider whether the behaviour of poisoned animals could cause them to be selectively predated.

It is well known that animals behaving oddly or moving sluggishly may be selectively predated. Rudebeck (1950) observed avian predators' hunting attempts and successes, and showed that over twice as many successful attacks were on abnormal or injured prey. Mech (1970) also found that the majority of ungulates killed by wolves were young, old or disabled. Wild brown rats show a tendency to move in contact with a vertical surface, known as thigmotaxis (Barnett 1963, Patrick and Laughlin 1934). We have observed poisoned rats on farms moving sluggishly in open areas in daylight, and this phenomenon was confirmed in discussions with experienced rodent control operatives. If there is a widespread change in behaviour after chronic poisoning, there could be selective predation by day-hunting predators. Kotler

et al. (1988) showed that barn owls had a higher predation rate on rodents foraging in the open.

Before any speculations can be made on the probability of secondary poisoning hazards of anticoagulants, it is necessary to discover what happens to a rat after a lethal dose of an anticoagulant. Does feeding cease and does the animal retreat to its burrow and die under cover? Harrison et al. (1988) quoted a figure of 4% of an original population found as carcasses above ground after a control programme. Fenn et al. (1987) stated that the majority die under cover, if this is so, then the hazard to scavengers at least is substantially reduced. But if the rats continue to feed, remaining active whilst behaving oddly, then predation could increase sharply.

In the cage experiment described below, behavioural changes following anticoagulant poisoning were recorded in individually caged rats and compared with normal behaviour before dosing. Untreated controls were run simultaneously to ensure that any changes were caused by the treatment and not any external conditions. Wild-caught rats were used as their behaviour would be more representative of farm rats than laboratory-bred strains.

Although cage trials give some insight into the behavioural response of wild rats to a lethal dose of anticoagulant rodenticide, the rats' use of available space cannot be determined. In particular, the phenomenon of thigmotaxis (the tendency to move in contact with a vertical surface rather than in the open) needs investigation in larger areas. Wild rats, once they leave the burrow, are subject to predation and one means of avoiding predators is to use covered pathways where possible to reach food supplies and harbourage (Barnett 1963). If poisoning affects the use of cover, perhaps leading to more time spent in open areas, then poisoned rats could be more vulnerable to predation.

A series of enclosure trials was planned to see if being in a social group affected the behavioural responses to anticoagulant poisoning observed in individually caged rats. An enclosure large enough to give open areas and with solid vertical sides plus covered pathways would be ideal. Patrick and Laughlin (1934) working with white rats found that those reared in glass-walled cages with continuous lighting showed a higher rate of exploratory behaviour, running out from the walls more readily than normally reared controls. But these were laboratory white rats selectively bred to lose many of their innate characteristics (Barnett 1963). The rats used in

the present work were bred from wild-caught parents, a compromise to provide animals with natural behaviour patterns whilst avoiding the inevitable fighting involved when strange wild rats are housed together in an enclosed space (Barnett 1963). Crozier (1928) indicated that thigmotactic behaviour is innate and not learned or linked to the conditions in which the rats were reared. The rats used in the present trials were reared in large wire mesh cages to ensure that as far as possible thigmotactic behaviour remained intact. By studying all aspects of behaviour after acclimation to the enclosures it would be possible to establish if such behaviour was present before dosing and if poisoning subsequently affected thigmotaxis. Taylor (1978) studying radiotracked wild rats found rats never traversed open areas in the daylight, and at night there was a tendency to follow the network of hedgerows on the study site.

Being in a group should provide a more natural simulation of the stresses that affect anticoagulant action on rats. Jaques and Hiebert (1972) showed that spontaneous haemorrhage from anticoagulant ingestion is a multicausative phenomenon and is greatly influenced and triggered by stress. Also slight injuries caused by interactions between animals may predispose them to bleeding, thus in turn affecting survival time which could be compared with the previous cage trial data.

In a large enclosure another aspect of feeding behaviour could be considered. Rats tend to feed in corners or near cover where possible (Barnett 1963, Meehan 1984) and this fact is emphasized in manuals dealing with their control. This behaviour relates to the avoidance of predation, as a rat feeding away from cover would be readily seen by potential predators. If two feeding stations were provided, one nearer to the nest box area but in the open and one further away but allowing feeding under cover, the rats' preference could be established. If they used a feeder with cover before treatment, poisoning might cause them to disregard safety and feed in the open. Or if suffering from physical ill effects, they might avoid travelling far and feed at the nearest point to their refuge area. This could add another dimension to the possible changes in the use of open areas and cover. Finally, knowledge of the amount of time spent in various activities in the dark and light before and after intoxication would be useful to link with previous findings.

The experiments described below were designed to answer the following questions about the behaviour of rats following intoxication with a lethal dose of an anticoagulant rodenticide:

1. does feeding and drinking continue normally?
2. does time spent out in the open increase or decrease?
3. is thigmotactic behaviour lost?

Since all anticoagulant rodenticides have the same physiological effects (Meehan 1984) we do not refer to any particular compound here. Differences in toxicity and metabolism of different compounds are important for other aspects of ecotoxicology (Smith et al. 1990) but not for pre-lethal effects on behaviour once a lethal dose has been ingested.

## CAGE STUDY

### Materials and Methods

Wild brown rats were caught in wire Fenn traps at a site

on the University Campus. Trapping was carried out at intervals between April and November 1988. Ten males and ten females were selected for the trials and randomly assigned to either an experimental or control role. As far as possible rats of similar weight were paired. The weight range of males was 130-375g and of females 140-300g. Rats less than 235g may be considered as juveniles and above that weight as adults (Hartley and Bishop 1979). The relatedness of individuals was unknown.

Cages for the trials were constructed of strong wire mesh and measured 56 x 34 x 16cm with a covered nest area 13 x 34 x 16cm. The nest area was separated from the main area of the cage by an aluminium partition with a single 6cm diameter hole for access. Hay was provided as nest material and the cages stood on metal trays lined with blue paper towel to allow food remains to be collected and faeces examined. The cages were painted with leadfree black matt paint in order to reduce reflection and improve the video image. A 12:12 light-dark regime was established with the dark span running from 10.00-22.00 in order that the rats could be watched during their active period. Light was provided by a 60 watt white bulb placed to the side of the cages and both cages received approximately equal illumination. A 40 watt red bulb was continuously lit to allow video recording during the dark period and to facilitate observations on the monitor screen. Rats were allowed to acclimatise for a week in the observation room (Syme et al. 1974) before seven days of feeding and drinking data were collected.

Trials were recorded by a surveillance camera with a wide angle, self adjusting lens placed above the two cages allowing both animals to be observed simultaneously. The camera was linked to a Panasonic 8066 VHS time-lapse video recorder set at 72hr and recording at 2 frames per second (125 frames/min.). Playing the tape back at normal speed had the effect of speeding up the action by a factor of twenty-four. Data for food and water consumption were obtained for seven days before dosing with rodenticide to provide a base line for later comparisons.

Video recording was carried out continuously from the seventh day of food recording until the death of the experimental animal. On the eighth day the normal wheat diet was replaced for the experimental rats by a commercial formulation of anticoagulant pellets. The control rats received an equivalent novel pelleted food (layers' pellets) which were selected to be similar in size and texture and without a pronounced smell. Controls were set up to check for possible moisture uptake. The novel foods were presented for 24hr and then replaced with the normal oven-dried wheat.

Ten trials were carried out using twenty animals, half controls and half experimentals. Trials with the male rats ran from April to July 1988 and with the females from September to December 1988. Thus five data sets for each sex were obtained and statistical analysis was carried out between and within the sex groups. All analysis was repeated for control animals.

### Results

In the period before dosing, there were no statistically significant differences between control and experimental animals or between sexes in any of the six behaviours or in food and water intake. There was no effect from the position of the individual ten trial. Males and females behaved in a similar

way and the timing of the trial had no effect on the result. Therefore any effects should be true effects of treatment and not initial differences between control and experimental groups.

Full details of the results and analysis are given in Cox (1991). The main results relevant to ecotoxicology can be summarised quite simply. Time to death after ingestion of a lethal dose of anticoagulants ranged from five to eight days in males (mean 5.8d) and from five to eleven days in females (mean 8.2d). Intake of both food and water declined rapidly in anticoagulant-treated animals, but not in control animals. Time spent in the nest box during the dark period increased from 55-60% pre-treatment to 90% the day before death. However, the time spent in the nest box during the light period fell from 85-90% pre-treatment to 62-67% the day before death. These changes were statistically significant and did not occur in control animals. Overall, time spent in the nest box increased following ingestion of anticoagulant but the normal activity rhythm was reversed such that animals spent more time out of the nest box during the light period.

## ENCLOSURE STUDY

### Materials and Methods

Twenty rats were used in the four trials, two males and three females in each group. A group consisted of litter mates born of wild-caught parents from Reading and Oxford. Their ages ranged between 100 and 150 days and the weights from 124 to 329g. They had been previously fed on SDS PCD pellets and this continued throughout the trials, except for the days when the rodenticide was presented. The rats had been reared in large wire mesh cages so thigmotactic behaviour should have been intact.

The two observation enclosures were erected adjacent to each other in a heated ventilated room with a mean temperature of 22°C and 15-20 air changes an hour. Enclosure walls were constructed from sheet aluminium and each measured 2.5 metres square with sides 1.2 metres high. Three 25cm square nest boxes made of metal lined with wood and filled with hay were provided in each enclosure. Water was supplied *ad lib* in two chick drinkers placed at opposite corners. Plywood tunnels 10cm square and 60cm long with clear plexiglass tops were placed along the sides of the enclosures, linking the drinkers to the nest boxes and a feeding station, which allowed the rats to eat under cover. A second feeding station was placed in an exposed position in the centre of the pen. Feed containers were plastic lidded buckets with holes cut at the base to allow pellets to fall freely onto a lipped metal tray.

Illumination was on a 12hr:12hr light-dark cycle with several 40 watt red light bulbs continuously lit to enable video recording to be carried out during the dark phase. The light phase ran from 07.00 to 19.00 in order that general movement in adjacent rooms coincided with the period of minimum rat activity. Water changing and feeding was carried out every two days to standardize disturbance and at all other times the room was closed.

Two trials were run simultaneously and each used a surveillance video camera with a wide angle, self-adjusting aperture lens, which enabled viewing of the entire pen area. The cameras were linked to time-lapse video cassette recorders in an adjacent room where monitors allowed observations to be made as necessary. Behaviour was recorded on 4 hour video

tapes at 2 frames/sec., 125/min., which when played back at normal speed had the effect of speeding up activity by a factor of 24.

A group of five rats was introduced into each of the two enclosures and left to acclimatise for 14 days with food pellets and water *ad lib* (Holler and Lefebvre 1981). Days 11-13 were recorded and 24hrs analysed to provide details of normal behaviour before poisoning occurred. On day 15, commercially formulated pellets containing an anticoagulant rodenticide were substituted for the PCD pellets in both containers. The rodenticide pellets were removed after 48 hrs. and replaced by normal pellets. Video recording continued from day 11 until the death of the last animal in the group. The position of dead animals was noted to determine if death occurred under cover or in the open. The enclosures and nest boxes were thoroughly cleaned before the introduction of the next two groups of rats.

### Results

A full presentation and analysis of results is given in Cox (1991). In one trial, two rats were found dead in the open during acclimation, but before videoing began. They were the two smallest in the group, but no sign of fighting was evident. By this stage it was inadvisable to introduce new animals because of the probability of attacks by residents on the newcomers (Barnett 1963), and the trial was continued with the three survivors.

All eighteen rats died within 120 hours of receiving the anticoagulant pellets, and three animals died within 68hr. The position of the bodies indicated that twelve rats died out in the open area of the enclosure and six died either in the nest box or under a tunnel.

Before dosing, the "normal" behaviour of the rats was to spend most of the light phase in the nest box, and of the time spent outside, significantly more was spent at the edges. At this stage most activity was in the dark and there was equal use of the two feeders. The normal light phase pattern of nest box usage of the group of rats mirrored the cage trials with a mean of 86% compared with 86.8%. The 29.5% of the dark spent in the nest box is much lower than that for the individuals in cages which averaged 63.5%. The difference could be because some rats in the group were ejected from nest boxes by resident animals, and certainly some rats were observed using the tunnels as a resting area. Subdominant animals may feed at less favoured times to avoid conflict with the dominants (Nott 1988).

When the animals were out, whether in the light or the dark, most time was spent at the edges or under cover rather than in the open. It appears that thigmotactic behaviour was innate in the rats used for the trials, and any changes later could be consequence of poisoning.

Forty-eight hours after presentation of the poison, changes were already occurring in the patterns of behaviour. A higher proportion of time in the dark phase was spent in the nest box in three out of the four trials, a mean of 48% compared with 29.5% before dosing. Rats were thus out and active for less time during the night. However, time in the nest box in the light decreased slightly in three of the four trials with a mean of 80.2% compared with 86% before dosing. Thus the rats were active for a larger percentage of time in the light than before and for less in the dark. Feeding in the light stopped after 60 hours in all cases, and in two trials no further

feeding occurred. At 48 hours there was a significant decrease in the amount of time spent at the edges and an increase in time spent in the open. Overall there was no significant difference in time spent in either place, but all trials showed more of the light phase spent in the open than at the edge. The trend to increased proportion of time spent at the feeder with cover continued. Thus after 48 hours the behaviour of the rats had already been affected by the poison and they were spending a greater proportion of the light hours out and active in open areas. The potential for being caught by a diurnal predator might consequently be increased especially with the external haemorrhaging which was observed in the cage trials.

By 72 hours after dosing rats were still using the edges significantly less and the open areas more than before, resulting in a similar amount of time spent in both. By this stage rats were observed standing still in the open for up to 20 minutes at a time. More of the dark was spent in the nest box than before which could imply less exposure to night-feeding predators such as owls and foxes. In two of the trials, more time was spent out and active in the light, a reversal of the "normal" pre-dose behaviour. There was no light phase feeding and there was dark phase feeding in only two trials. So the rats were not emerging in the light to feed and drinking only occurred on one occasion in the light.

By 96 hours after presentation of the poison, seven of the eighteen rats were dead. Death occurred much sooner than in the cage trials, presumably because behavioural interactions hastened haemorrhage as previously detailed. Feeding occurred in one trial and drinking in two others during this day (96-120 hours after presentation of anticoagulant), mainly in the dark. Use of the edges continued at a lower level than before dosing, but use of open areas was comparable. Again more of the dark phase was spent in the nest box, but time inside in the light was unchanged. Activity patterns were reversed during the last 24 hours in three of the trials with rats active for more time in the light than the dark. When time out of the nest box spent either in the open or at the edges only is considered there was an upward trend from dose day onwards in the use of the open areas of the enclosure. Before dosing the open area was used for a mean of 15.8% (S.E. 2.35) but by 96 hours after this figure had increased to 79.4% (S.E. 10.9).

## DISCUSSION

In the cage experiments described here, rats with high anticoagulant residues were in the open behaving normally in respect of the activities measured, forty-eight hours after intoxication. By this stage, six of the ten animals were bleeding from the nose and paws and some showed sluggish reactions to touch. Whilst feeding and moving around their territory trails of blood would be left which could attract predators. Slower reactions might mean that they could be caught more easily. Thus although their time in the open and hence availability to predators was unchanged from before poisoning, the presence of blood and the different startle response, from bolt to freeze, could increase their vulnerability. With feeding behaviour unchanged, already poisoned dominant rats could exclude less dominant animals from the poisoned food source. As the dominant rats are usually the largest (Barnett 1958a, Nott 1988, P. Smith, pers. comm.), there could be a size effect on patterns of mortality when farm rodent

control was carried out.

During the last 24 hours before death, all behaviours except time spent inactive had changed significantly, with time in the nest box increasing and the others decreasing with the result that animals would be less available to predators hunting over open areas. Although the time spent out of the nest box was less on the final day, all animals were out for at least part of the twenty-four hours leading to death. By this stage all exhibited symptoms of haemorrhaging with the males staggering and all animals freezing rather than bolting to cover when alarmed. By appearing in daylight with these symptoms rats may be easily taken by diurnal predators such as kestrels, buzzards or short eared owls. In urban areas rats make up nearly 20% of the kestrels' diet (Cramp and Simmons 1980). Buzzards will take rodents active by day and Shawyer (1985) described an incident in Switzerland in which one hundred and eighty-five buzzards were found dead after a second generation anticoagulant was used to control water voles. Vernon (1972) listed *R. norvegicus* as a major prey item of the short-eared owl in Cambridgeshire, amounting to 83% of total prey remains in one hundred and sixty-seven pellet samples. Foxes and badgers will take rats opportunistically and, although rats do not form a large part of their diet, Day (1968) acknowledges rats as an important prey source for mustelids when locally abundant. It is possible that the presence of a large number of dying rats in the small area of a farmyard might attract both nocturnal and diurnal predators with a consequent potential for secondary poisoning effects.

Secondary poisoning could also occur if dead rats were found by scavenging animals. Half of the caged rats did not die under cover and appeared to move deliberately out from the shelter of the nest box to the open area of the cage. They were not necessarily feeding or drinking, but some just lay on the exposed floor of the cage. If this behaviour is not just a peculiarity of caged wild rats, then in the field the moribund animals would be easily seen and taken by scavengers. Scavenging efficiency would appear to be high, with three-quarters of carcasses removed in three days or less (Balcomb 1986, Heinz et al. 1979, Phillipson and Wood 1976).

The enclosure studies provided additional information on use of space by animals housed in groups. Observations showed rats emerging from nest boxes, staggering into the centre of the pen and sitting motionless. Others climbed onto the roof of nest boxes or food containers and remained there, sometimes in groups of two or three. The motivation for this behaviour is not obvious and seems not to have been previously documented. Farag (1982) discussing the effects of sublethal doses of anticoagulants on reproduction, mentioned "carelessness" which he did not define further, and loss of appetite at three days. Desheesh (1983) looked only at "hesitancy" and "refusal time" when considering the effects of poisoning. He mentioned the onset of illness (undefined) and "drastic behaviour changes" but did not describe them. Godfrey (1984), describing wallabies poisoned with an anticoagulant, mentioned only lassitude and anorexia. It may be that some sort of brain damage had occurred as a result of the poisoning here. *Post mortem* examination of anticoagulated rats often reveals the presence of cerebral haemorrhages which could account for such aberrant behaviour. If this behaviour happened in the wild after control treatments, then rats could be easy prey for nocturnal or diurnal predators. The

findings confirm the change in startle response from bolt to freeze observed in the cage studies, with rats remaining motionless when an observer entered the pen. Even climbing into the enclosure to renew the water had little effect, when previously the rats would have bolted to the nest boxes or covered tunnels.

In the field, Harrison et al. (1988) and Fenn et al. (1987) did not find many dead rats above ground, though Gemmeke (1990) in his experiments on various caged rodent species showed that animals poisoned by anticoagulant rodenticides died as often above ground as below. It is possible that rats behave in a different way under natural conditions, or it could be that in the field predators and scavengers remove carcasses rapidly. It is also possible that in farmyards and hedgerows there are many elements of cover that an enclosure does not provide. Rats may crawl under stacks, corrugated iron or machinery and not be found, or they may remain in their burrows whilst suffering the severe physical effects of poisoning. Their emergence in the enclosure trials here does not seem to be related to feeding or drinking in the latter stages of intoxication. In fact pellet hoarding was evident in most nest boxes and emergence to feed would have been unnecessary.

Why the light/dark activity patterns changed is also not evident. If the rats were staggering or reacting abnormally, it might be thought that keeping to cover and only moving in the dark would be optimal to minimize risks of predation. It is possible that blindness occurred, or that internal biorhythms (Broom 1979) which enable animals to wake and become active at the safest time were also affected by the poison.

Whatever the cause, appearing in the light in open areas and sitting motionless for considerable lengths of time could increase the rats' liability to predation. Gemmeke (1990) came to similar conclusions from his studies on the effects of rodenticides on various rodent species as his animals lost "their shyness and partly their nocturnal disposition." Thus there is indication of a potential secondary poisoning threat to diurnal predators. Nocturnal predators might encounter intoxicated rats for a shorter time period, but rats would be more vulnerable during periods in the open. The presence of a large number of such moribund animals in a limited area following a farm control programme could also facilitate the development of a "search image" (Dawkins 1971, Tinbergen and Drent 1980) and again might increase the number of poisoned animals taken by an individual predator.

If, as indicated here, a high proportion of poisoned rats die in the open, then again there would be an increased risk to scavengers. Bodies may be collected and "cached", behaviour frequently recorded in foxes (MacDonald 1987) and mustelids (King 1989). Caching could lead to the death of the scavenger collecting the carcasses as well as other scavengers that found the cache later. Alternatively, if the prey were taken gradually over a period of time, sublethal effects might occur which could have possible effects on the breeding of avian species.

In summary, our results indicate that pre-lethal effects of anticoagulant rodenticides are likely to alter the exposure of predators and scavengers to rodenticides. Diurnal predators may be exposed more than nocturnal predators. However, we emphasise the need to extend these detailed studies of behaviour to the field using indirect techniques such as radio-tracking and remote videorecording as well as direct observation.

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